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EAST AFRICA PROTECTORATE.

ANNUAL REPORT

OF THE

Veterinary Pathologist

FOR THE YEAR

1909-10.

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ANNUAL REPORT
OF THE
VETERINARY PATHOLOGIST, FOR THE YEAR 1909-1910.

NAIROBI,
June 30th, 1910.

To

THE CHIEF VETERINARY OFFICER,
NAIROBI.

Sir,

I have the honour to submit herewith my first Annual Report as Veterinary Pathologist. The period covered in this report extends from the time of my arrival in the Protectorate up to the present date. It is only within the past two or three months that certain results have been obtained and as these appear to be of some interest and importance to stock owners it is considered advisable to embrace them here, rather than to close our record on March 31st, and delay their publication until the next Annual Report.

I arrived in this Protectorate on August the 28th, 1909. During my visit here in February 1909, the question of a Laboratory was discussed, plans and estimates obtained, and a suitable site was selected on a corner of the Kabete Government Farm. On leaving England I was informed that orders had been issued by cable authorising the immediate construction of this building and that I should find it almost ready for occupation on arrival. I regret that even to-day the Laboratory is not completed; but I sincerely trust that it will be sufficiently advanced for some work to be commenced there within the next two months. I have consequently spent most of my time on tour, and in conducting experiments in the field. This experience has been of advantage in enabling me to obtain a first-hand insight into the local conditions affecting stock raising, and to appreciate, in some degree, the many factors underlying the application of scientific methods to practice, and cannot altogether be considered wasted.

It may at first sight appear somewhat disconcerting to read the list of the many serious diseases which have been encountered during the period under review, and it might be held to indicate that East Africa is not well suited to stock. Such an opinion could only be formed by one not conversant with the country, the nature of the diseases in question and rational means for their eradicatio

There appears every ground for believing that most of th diseases affecting stock now known here are capable of preven ative or curative treatment.

The new Laboratory has been designed with the object an intention of entering into all the ordinary fields of enquiry regard ing stock diseases. It has of late become increasingly apparen that serum and vaccine production will constitute a maj portion of its function. The adequate development of this branc will require the installation of additional apparatus, and the cons truction of more out-buildings, the details of which will be o already have been laid before you.

It appears advisable to briefly review the present position regarding diseases in a general manner, and to reserve for appendices the more detailed observations.

Trypanosomiasis, which has been detected in 53 animals during the year, has not yet given way to science in a manner capable of practical application. It is not, however, a disease which seriously affects that portion of the Protectorate given up to stock raising. The cases which have occurred are mainly in animals traded from the territories to our north, or in those which have been taken into areas known to be dangerous. It is conceivable, however, that in the future this disease may affect more seriously some of those planters in the Coast Belt who are dependent upon oxen for transport.

Coccidiosis is another disease of which we know little from a preventative point of view. This disease will be discussed more in detail in Appendix iii; here it will suffice to say that it has been diagnosed at Meru, Kisumu, Kericho, Naivasha, Lamu and on the Island of the Ukerewe in German East Africa. It was also in all probability present in the cases of disease which were observed by yourself and also by Theiler on his visit here, the blood of which was non-infective to clean animals. The disease engendered by this parasite is, so far as my observations go, almost identical with Rinderpest. It is a disease known in certain Cantons of Switzerland, where it recurs on particular farms or in particular districts, and accounts for a mortality of about 10 to 20% of infected stock. The symptoms noted by the Swiss writers, Guillebeau and Hess, are quite comparable with those seen in East Africa, and the similarity is indicated by the local names "Red dysentery" or "Bloody diarrhœa". I have had but small opportunity for observing the life cycle of the coccidium; such stages as have been noted are identical with those described in Europe: we must therefore assume that the ripe parasite or oocyst may contaminate a pasture for a long time. Medicinal and hygienic treatment has proved efficacious in combating the mortality, but a considerable amount of research is necessary before we can hope to be in a position to control the morbidity. In this enquiry we have been severely handicapped by the introduction of a second disease which, though most closely resembling coccidiosis, is due to a distinct cause. There can be no question but that in some instances these two conditions have been running side by side, and it is therefore, impossible to separate the evidence relating to the one from that relating to the other, and it will be imperative for us to be able to reproduce the uncontaminated coccidial disease before any exact study can be made.

No detailed research work has been conducted on the disease due to the genus *Piroplasma*, but so far as this affects canines, it appears probable, and my experience coincides, that the drug Trypanblau introduced by Nuttall and Hadwen, has a pronounced beneficial effect when scientifically employed. I have had no opportunity of noting its effect in cattle, horses or sheep affected with other piroplasmoses, but I understand the indications have been favourable. In the disease due to the allied parasite *Theileria* or *Piroplasma parva*, of East Coast Fever, we are, I believe, in a position to throw some light. On my arrival I was much struck by the scepticism of otherwise intelligent stock owners with regard to this disease, and finding that no records existed in print, it has been necessary to repeat a number of experiments to prove that the disease of this country is apparently the same as that in South Africa. In Appendix ii, will be found records showing that the course, duration, symptoms and post-mortem appearances in the two countries are identical, and that the same causal parasite and the peculiar form known as Koch's body (Blue Body) is also found. The experiments by means of ticks were, unfortunately, delayed owing to my enforced absence, but the cases recorded justify, in my opinion, the view that the disease known locally as East Coast Fever, is identical with that which decimated Southern Rhodesia and the Transvaal, and which is even now causing most severe losses in Natal. Any differences must be ascribed to the greater immunity enjoyed by some native cattle.

As a routine system the diagnosis has been made on the occurence of Koch's bodies, which in my present opinion, may be held to be pathognomonic of this disease. If this be accepted, we must admit the occurence of East Coast Fever in Egypt, Trans-Caucasia, India, German and Portuguese East Africa. In German East Africa it has been shown to be endemic in certain districts, and some observations have been made to ascertain whether this may not be the case here. Our experience goes to show that in certain localities, at least, of the Kisii and possibly also of the Nandi Countries, the adult cattle are immune. The explanation of this is probably that the calves become infected soon after birth, and that in them the mortality is not very severe. Bruce and his collaborators have stated that a disease common in calves in Uganda known as "Amakebe" is East Coast Fever. This same, or a clinically similar condition, is frequent in many parts of the Protectorate, but it seems undeniable that all cases of "Amakebe" here are not Coast Fever. In the Kisii, Nandi and possibly also the Wakamba districts many may be associated with Koch's bodies. If this be the case it is an open question whether the policy of eradication, save in definite areas, might not with advantage give place to one which would tend to develope an immune stock. So far as the question of transport oxen within the areas which it is desirable to clean is concerned, I can at present foresee no difficulty in obtaining a sufficient number of oxen, which on testing are proved immune, to enable transport to proceed.

I trust experiments will be instituted immediately to ascertain whether calves born of highly susceptible parents may not be artificially infected at an early age with only a loss which would be compensated by the enhanced value and usefulness of the recovered and consequently immune stock.

Another disease of a tick-borne nature has caused rather serious inconvenience amongst the pure-bred imported cattle at the Naivasha Government Farm. The parasite concerned, the Marginal Point, recently named by Theiler *Anaplasma marginale*, is transmitted by the Blue Tick, *Boophilus decoloratus*. The same parasite has also been observed in specimens coming from other parts of the Protectorate. The disease is in many cases favourably influenced by medicinal treatment, but it is one which requires investigation to ascertain if the introduction of pure-bred cattle can be continued into the affected districts without danger.

The other principal diseases which affect the live-stock of East Africa belong to that class due to an ultra-microscopic organism. We have not yet actually shown that the causal organism of the disease which has come in from the North and which has been confused with the non-inoculable Gastro-enteritis

(Coccidiosis), is ultra-microscopic. But there is no hesitation in saying that the symptoms and post-mortem appearances of both naturally acquired cases and those conveyed by the inoculation of small quantities of blood are identical with Rinderpest.

The evidence available is that this disease entered East Africa from the Boran Country to our North, and coming, as it did, at a time when the non-inoculable Gastro-enteritis was still fresh in the minds of stock owners, it is small wonder that the two should have been confused. A virulent outbreak among the work oxen on the wheat farms at Njoro was the first to arouse suspicion. Enquiry has shown that from the causative standpoint the two diseases are in no wise related, and that an attack of Coccidiosis confers no immunity to an inoculation with the second disease. As an inoculable disease we have reason to hope that a serum may be manufactured which will possess protective properties. There appears to be every justification for this view, and the experiments conducted up to date indicate most hopefully that anti-Rinderpest serum is efficacious. In Appendix IV are detailed some of the experiments which have so far been conducted with the serum obtained from the Transvaal Government. Even allowing 50% for loss of potency owing to age and bad storage we can, I hope, place about 7,000 doses among cattle owners. By the time this supply is exhausted we should have a sufficiency of locally prepared serum, and by the adoption of such system as seems best adapted to local requirements there does not seem any grave objection to a belief that this disease may be eradicated within the next two years. The treated cattle will then possess immunity which will ensure their safety for such a time as may permit of an organisation being instituted to prevent a further introduction from the pathologically unknown territories of the North.

It does not appear likely that we have much to fear from the South, but on the North and West we are justified in maintaining an energetic quarantine until these countries have been thoroughly surveyed by a veterinary staff.

I should like to urge that every effort be made to stamp out this disease, the collective losses from which probably amount to over ten thousand head. The establishment of a serum making station in conjunction with the Laboratory, on completion, would cost but a fraction of what the saving by immunisation will be. I consider the present time the most opportune; for it is within the bounds of possibility that with each successive outbreak the virulence and the mortality will increase.

Horse sickness has been responsible for several deaths within the period under review. Serum has been obtained from the Transvaal Laboratories, but we are not yet in a position to express any opinion regarding its efficacy towards the strains existing here.

I was enabled to observe an outbreak of a disease resembling Blue Tongue in sheep on the Naivasha Government Farm in February last. At that time practically no rain had fallen and the position of this farm makes it difficult to believe, *a priori* that any insect commonly associated with damp low-lying and swamp lands should have been concerned in its transmission. We were able to prove the inoculability of the disease into merino sheep, but the experiments in native and half-bred sheep were negative. I think this is a disease which might receive attention and for which serum might be prepared. Although the losses are as yet small, it is conceivable that the virulence may increase. In any case, it would encourage the importation and breeding of more valuable stock if sheep owners could be assured that their animals might be immunised.

Swine fever is another disease which is due to an ultra-microscopic organism. A severe outbreak on two adjoining farms near Nairobi has served us with material for commencing a study on the condition as it occurs here. If we can satisfy ourselves that the inoculations are positive, and that the serum advocated by the American writers is of value, it is obvious that the production of anti-Swine-Fever Serum would be of great benefit to settlers, many of whom regard swine as a very valuable asset to the Protectorate. These experiments were only recently instituted and no results are yet available.

I have not had any opportunity of studying the various forms of disease due to intestinal or pulmonary worms, which have at times accounted for serious loss to stock owners. It is, however worthy of notice that in three aged transport oxen dead of Coccidiosis at Kisumu, I found "Bilharzia" worms present. The species I believe to be *Schistosomum bovis*, the Egyptian form, which is characterised by its spindle shapped egg. Adult females were obtained from the mesenteric veins and the ova were found in the rectum. I had great opportunity for observing the effect of parasites of the same genus in India, but was unable to adduce any evidence to show that they produce ill results at all comparable to those due to the human form, *Sch. hæmatobia.*

Among the pathological conditions as yet unknown to us must be mentioned N'garuti. The Masai name is applied to a fairly common disease in sheep, and, according to flock owners, accounts for a larger mortality than any other ovine disorder. Near Elmenteita I had an opportunity of seeing four cases which were regarded as this disease by owners and sheperds. The inoculations made from these animals did not reproduce the disease, thus lending colour to a widely held view that a poisonous plant is responsible. It is hoped that further observations will be possible during the year, but as the death of the patient is usually the first indication of trouble the difficulties to be overcome are great.

Another most interesting condition in sheep has recently been observed, and fortunately it is limited, so far as is known, to one farm. Last lambing season the owners lost 82 lambs out of 240 from an enlargement of the thyroid glands. In every case the lamb was born with this deformity, and in only three instances did the lamb live for more than a few days after birth, usually they died within a few hours. I am informed that the three which recovered now show no sign of enlargement. It would seem to be a congenital defect, but from the available evidence, neither the male nor the female stock could be blamed, both having previously bred healthy sheep. The owners have kindly promised to regulate the mating next season in accordance with a scheme, which will be drawn up, having for its object the elucidation which class of animal or which individual is mainly implicated, for it is difficult to conceive that this disease can influenced by any local conditions.

I forward herewith four Appendices, setting out in greater detail the year's work.

Appendix i	Showing the routine examination at the Laboratory.
Appendix ii	East Coast Fever.
Appendix iii	Coccidiosis.
Appendix iv	Rinderpest.

I have the honour to be,

Sir,

Your obedient servant,

R. EUSTACE MONTGOMERY,

Veterinary Pathologist.

APPENDIX I.

Microscopic examination of Slides sent to the Laboratory, April 1st 1909—March 31st 1910.

1. EQUINES.

A. HORSES.

Trypanosomata	14	
Piroplasmata	12	
Epizootic lymphangitis	3	
Ulcerative lymphangitis	17	
Anæmia	1	
Negative	117	
		164

B. MULES.

Trypanosomata	5	
Piroplasmata	11	
Epizootic lymphangitis	2	
Ulcerative lymphangitis	7	
Anæmia	2	
Negative	206	
		233

C. DONKEYS.

Trypanosomata	7	
Piroplasmata	10	
Anæmia	2	
Negative	22	
		41

2. CATTLE.

Trypanosomata	27	
Koch's bodies (East Coast Fever)	211	
Small piroplasmata=*P. mutans*—*T. parva* ? ? ?	103	
Babesia bigemina (Redwater)	30	
Marginal points *(Anaplasma marginale)*	3	
Anæmia	33	
Spirochæta	1	
Sarcosporidiosis	1	
Coccidia (*Eimeria stiedæ*)	3	
Anthrax	1	
Negative	1,343	
		1,756

3. SHEEP.

Anæmia	3	
Anthrax	1	
Negative	39	
		43

Carry forward... 2,237

(APPENDIX I.)

	Brought forward ...		2,237
4.	GOATS.		
	Negative	14	14
5.	CAMELS.		
	Trypanosomata	3	
	Negative	10	
			13
6.	PIGS.		
	Anæmia	2	
	Negative	9	
7.	DOGS		
	Trypanosomata	1	
	Piroplasmata	42	
	Anæmia	32	
	Negative	158	
			233
8.	FOWLS.		
	Tuberculosis	1	
	Bipolar bacteria (Fowl cholera?) ...	5	
	Negative	16	
			22
9.	TURKEY.		
	Negative	2	
			2
10.	OSTRICH.		
	Negative	1	
			1
11.	WILD ANIMALS		
	Negative		
	Eland	1	
	Kongoni	3	
	Zebra	1	
	Thompson's Gazelle	2	
	Dik-Dik	1	
			8
	Halteridium		
	Marabou Stork	1	
			1
	Total Examinations ...		2,542

NOTE.—These figures refer solely to slides and material forwarded to the Laboratory; they do not include any examinations made when on tour. Dr. Ross, the Bacteriologist of the Medical Department, has most kindly controlled all positive diagnoses made during my absence from Nairobi.

APPENDIX II.

East Coast Fever (Tropical Piroplasmosis).

Until we have been able to study this disease as it occurs among the different breeds of cattle or in various district of this Protectorate, and until such time as we are in a position to reproduce the disease experimentally by the means of ticks, the genesis of which is known, we must regard the question as incomplete from a scientific standpoint.

The reports of the Chief Veterinary Officer for the years 1907-1908 and 1908-1909 contain all the information available on this subject to the date of my arrival. From them it is apparent that considerable immunity exists in some cattle, but the type, whether inherited or acquired, and the geographical distribution of immune stock is, to the present time, not finally known.

I was able to devote myself to this desease for two weeks in January and again since about the middle of March. The work was carried out on the land of Mr. T. Russell near the Kamiti River, some 13 miles from Nairobi, to whom the Department is much indebted for the facilities offered. On the land in question 121 head of native owned cattle died between the months of November 1909 and February 1910, the remainder of the herd, 49, being apparently saved by passage through a temperature camp and movement to a clean area. During my absence in German East Africa, the month of February and the first eleven days of March, the experimental animals were under the charge of Stock Inspector Thorne, who carefully recorded the temperatures and general appearences.

It is considered adviseable to include in this report the result of such experiments as were in progress but not completed at the termination of the Official Year—March 31st.

I. To observe the Period of Incubation and Duration of the Disease.

1. Two half-bred calves, Nos. 24 and 25, born on the Government Farm Kabete, were exposed, together with their mothers, cows No. 23 and 26, on February 1st, 1910.

The morning temperature of the calves rose to exceed 103·4° on the 16th and 19th days, and the animals died on the 29th and 28th days respectively. The mothers have shown no disturbance in their temperatures to the 75th day.

2. Eleven half-bred bullocks, Nos. 31 to 41, about 18 months old, bred near Naivasha, an area free of tropical piroplasmosis, were exposed on February 14th, 1910.

The temperature of five rose on the eleventh day, of four on the twelfth, one on the fourteenth and one on the sixteenth day—an average incubation period of 12.09 days. Two died on the twenty second day, three on the twenty fifth, one on the twenty sixth, two on the twenty seventh and three on the thirtieth day, giving an average duration of 14.18 days for the disease : a total of 26.27 days from exposure.

Six of these animals were subjected to a preventative, and later a rative treatment, which was regarded by certain stock owners as of value this disease. There was no modification of the disease in these animals.

3. Ten quarter-bed bullocks of about 12 months old were exposed at fferent times between March 16 and March 28th, of which Nos. 49, 53, d 54 served as controls. The temperatures rose on the 14th, 15th and th days (average 14 days) and the animals died on the 31st, 31st and nd days of disease (average 14 days).

These three experiments, comprising sixteen susceptible animals from ree separate localities, give an average incubative period of 13.12 days, d an average duration of 13.74, or a total average of 26.86 days from posure on the infected land up to death.

II. SYMPTOMS AND PATHOLOGICAL LESIONS NOTED.

During the course of the disease the only characteristic symptom is the reat elevation in the morning temperature which, until the last few days, arely falls below 104° (40° C) after the initial rise, and has been recorded s high as 107.3° F. (41.8° C.) In this country, as in certain other tropical ountries, the morning temperature of apparently healthy bovines ranges sually between 101° and 102° (38.4° to 38.8° C.) the evening temperature, n the other hand, being more inconstant: after the usual grazing it may e said to average about 104° (40° C.) in most animals; some have, owever, come under my notice which rarely register less than 105° 40.5° C.). For this reason stress is laid upon the temperature registered efore 8 a.m., and, for the administration of a temperature camp, a rise to 03° (39.5° C.) warrants removal and segregation. In estimating the eriod of incubation, the initial rise to this point is held to constitute the nset of disease. Further than this elevation of temperature no symptoms re constant. In our experimental animals, however, as well as in one utbreak I was able to observe, a marked enlargement in the superficial ymphatic glands was observable.

Practically all bovines in this country, as well as in Northern Rhodesia, possess precrural and prescapular lymphatic glands of considerably larger size than is usual in England—each being frequently as much as two inches in length and one to one and a half in diameter—and, especially the precrural, are visible in the standing animal. During the course of this disease the prescapular may attain a relatively enormous size, and be easily seen projecting from beneath the shoulder joint. They have been found to measure as much as four inches in length, nearly three in breadth and two in thickness, being sufficient to fill the hand when held for gland puncture. The most noticeable gland, however, is a small one lying just below the base of the ear, on the edge of the jaw bone and situated beneath the parotid gland. A few only of the apparently normal cattle, chiefly young stock, show any enlargement of this gland, but during the disease it not infrequently attains the size of a small orange. This generalised superficial glandular enlargement commences soon after the initial thermal rise, attains its maximum about the fifth to seventh day, from which time the size decreases, and at death they are but little larger than before the attack.

There is no acute inflammation of the glands during enlargement : pain on manipulation is usually absent.

The affected animals feed well and maintain their condition until within two or three days of death. Despite a high temperature, not infrequently 108° in the evening, the ox will chew his cud and present an almost normal appearance. During the final days anorexia is common and emaciation rapid.

In six of the half-bred animals brought from Naivasha, hæmoglobinuria was manifested late in the disease: this has not been noticed in those animals which were bred in the neighbourhood of the experimental area.

On autopsy the prevailing lesions are œdema, ulceration of the abomasum and infarction of the kidneys. These are by no means constant and at some post-mortem examinations no characteristic macroscopic lesions may be seen. There is usually a generalised enlargement of the interna lymphatic glands—the larger ones being œdematous and the smaller somewhat hæmorrhagic in addition. A few cases present a marked œdema of the lungs—the trachea and bronchi being filled with forth, and subpleural hæmorrhages are occasionaly seen.

III. Efforts to reproduce the Disease by Inoculation.

On January 8th 1910, two naturally infected native calves, each showing about 75% of the corpuscles invaded by *T. parva*, were bled; and their blood, in citrate solution, inoculated into two healthy bullocks, Nos. 3 and 4, coming from Elmenteita: each received 50.0 c.c. subcutaneously. Up to March 31st there has been no rise in temperature and the animals remain perfectly normal.

No. 6, a companion bullock from Elmenteita, was inoculated intravenously on March 16th with 15·0 c.c. blood from an experimental animal dead from this disease. There has been no reaction to this inoculation.

Having in view the negative results obtained by Theiler and Nuttall with the subcutaneous or intravenous inoculation of blood or extract of organs, attempts have been made to reproduce the disease by the inoculation of Koch's bodies into organs wherein they are normally found. The connection of these bodies with *T. parva* will be discussed later, but it will be remembered that Mayer has succeeded in reproducing them in two animals into whose peritoneal cavities the spleen or portions of a spleen from cases recently dead from East Coast Fever had been placed.

Seven young bullocks of about 15 to 18 months old were obtained from a clean farm and maintained in a camp free from all infection.

Nos. 66 and 67 received into the substance of the prescapular gland on April 24th, 5·0 c.c. of an emulsion made from the glands of an experimental animal, No. 58, showing Koch's bodies.

Nos. 70 and 74 were each inoculated intraperitoneally on April 24th 1910 with 5·0 c.c. of the same emulsion.

Nos. 64, 73 and 77 served as controls.

None of the inoculated animals, 66, 67, 70 and 74, showed any disturbance in temperature or general health as a result of the inoculation and they were exposed to the infected area, together with the controls, on June 9th.

Of the controls, No. 64 died six days after the experiment was instituted, without showing any lesions of Coast Fever. The remaining two both developed a typical attack of Coast Fever and died on the 28th and 25th days after exposure.

Both Nos. 66 and 67 showed a very slight rise in temperature (102·6° in the morning) for five days after injection, otherwise they remained normal. No. 66 commenced a reaction on the twelfth day after exposure to the infected area, and died on the twenty fifth day No. 67 showed a slight reaction on the 13th, 14th and 15th days from exposure, and again commencing on the twenty second day and lasting for eight days. From this time the temperature has been normal and the animal appears to have recovered : 51st day.

No. 70 commenced a temperature reaction on the 16th day after exposure and died on the thirty third day of Coast Fever.

No. 74 commenced a reaction on the 20th day, which lasted for five days. The temperature then fell to normal and the animal appears to have recovered.

IV. THE TRANSMISSION OF EAST COAST FEVER IN NATURE.

Lounsbury and Theiler having shown that the genus *Rhipicephalus* (Neumann) is mainly if not exclusively concerned in the transmission of East Coast Fever in South Africa, it is necessary for us to ascertain the species occurring in this Protectorate, and their distribution.

I have obtained the following species of Ixodidæ in the vicinity of Nairobi and Kamiti :—

Margaropus annulatus, var. *decoloratus*.
Rhipicephalus appendiculatus.
Rh. sanguineus
Rh. pulchellus.
Rh. evertsi.
Rh. simus.

Two other species of *Rhipicephalus* allied to this last named.

Hæmaphysalis leachii.
Amblyomma variegatum.

With the possible exception of the *Rh. pulchellus*, which is more prevalent on the Athi and Kapiti Plains, the other species of this genus appear to have a wide distribution in the Highlands, and Mr. Anderson, the Entomologist, informs me that Professor Nuttall has determined *Rh. capensis* as occurring in this Protectorate also. We appear, therefore, to have all five species which have been shown capable of transmission and in addition *Rh. sanguineus*, *Rh. pulchellus* and two other species which will most possibly be found equally capable.

These species are now being reared, and the results of experiments undertaken to demonstrate their powers of transmission will be recorded in due course.

The figures do not include the routine examinations of spleen smears made from all oxen killed in the Nairobi Slaughter House, to which reference will be made later; nor many of those forwarded for diagnosis and concerning which the details are unknown.

In no case have Koch's bodies been found when Coast Fever could not be suspected and was not subsequently shown to be in the vicinity (the one Lumbwa case).

During my visit to the farm of Mr. Russell on the Kamiti, I had an opportunity of comparing the conditions in both sick and healthy side by side. On this land 180 native cattle were grazing up to November 1909 when the disease first appeared. In December all surviving animals were put into a Temperature Camp, and at the time of my visit in January, 71 were still alive, of which 59 had shown no sign of disease (temperature reaction) and 12 were segregated as being affected. Thirty five out of the fifty nine were gland punctured with a negative result in every case: while all twelve affected showed numerous Koch's bodies in their glands, and the Laboratory returns show that they had been found in all animals which had died on this land.

Whilst acting as Bacteriologist, Dr. Small instituted a system whereby a spleen smear was forwarded by the Meat Inspector from each ox killed in the Nairobi Slaughter House. During the Official Year 1909-1910, 733 such slides have been received in the Laboratory, and in 9=1.22% Koch's bodies have been demonstrated.

Nairobi being an infected area and its meat supply being largely drawn from the Kyambu area, this small number can be readily accounted for.

Nearly all the 54 negative slides of cattle in the Kenya and Naivasha Provinces were made from animals affected with Coccidiosis or its sequelæ. Amongst the other forms of disease from which slides have been examined with negative results are Piroplasmosis (*B. bigemina* and *B. mutans*), Trypanosomiasis, and various bacterial or parasitic (Helminthiases) infections. In addition, slides of all the domestic animals and a good variety of game have been examined at various times; Ross's finding in a slide which was forwarded as coming from a hartebeest (*Bubalis Cokei*) is the only positive record.

In the glands of a considerable number of apparently healthy calves may be found a blue staining body in size and appearance not unlike some forms of Koch's bodies, and which also frequently contains dots of chromatin. Side by side with Koch's bodies they are not difficult to distinguish, the chromatin being very irregular in size and distribution and stains rather bright red with the Romanowsky modifications. In Koch's bodies, on the other hand, the chromatin dots in any one cell are usually of approximately equal size and have a fairly regular distribution: further they acquire a deeper purple tint on staining.

It is conceivable that in some few instances these cells may have been mistaken for Koch's bodies; and it is possible that they afford an explanation of M. Mayer's statement.

So far, therefore, as this Protectorate is concerned I consider the evidence that has been obtained indicates in a very marked manner that Koch's bodies are not to be found in any bovine, except those suffering from Coast Fever: and that for administrative purposes their detection should be accepted as proof of this disease until a contrary explanation be forthcoming.

II. To note their association with the disease.

For this observation ten half or quarter-bred animals—Nos. 49 to 58—were exposed to the infected area : one did not acquire the disease and one, which was submitted to treatment, never showed Koch's bodies. Eight were therefore available for examination, which was made daily, so far as possible, by gland puncture

The following table shows the day after exposure when the official rise in temperature took place, and the day when Koch's bodies were first seen in the glands :—

Experiment No.	Day after exposure when temperature rose (103° morning.)	Day after exposure when Koch's bodies first seen.
49	14	16
50	15	18
51	15	18
53	15	16
54	13	13
56	14	17
57	14	17 Not examined on 16th day.
58	14	14

Note.—In both Nos. 54 and 58, in which Koch's bodies were seen on the same day as the "official" rise, the temperature on the day previous was higher than normal.

In six—Nos. 49, 51, 53, 54, 56 and 58—Koch's bodies were present at every examination to death, and on autopsy were found in all internal organs.

Cases 52 and 55 at no time showed these bodies, and are now alive.

Case No. 57 was an experimental animal to note the influence of serum from a recovered ox. After four days fever, the temperature fell and has remained practically normal since the 19th day after exposure. Definite Koch's bodies were only seen on two days, the first being the third after the official rise, and on the following day. For two days afterwards cells which might be held to have relation to Koch's bodies were seen: they were, however, not of the form which would be accepted as diagnostic, and require further study. This animal is still alive.

Case No. 50 is of great interest, and shows that the absence of Koch's bodies does not negative a diagnosis of Coast Fever.

This animal was exposed on March 19th and the official rise occurred on the 15th day. The temperature remained elevated until April 18th, the 30th day from exposure; only on two mornings was it below 105° (40.5C.) On the 31st day, the temperature rapidly fell and the animal died on the 34th: Koch's bodies were first seen on the third day after the official rise and were present at each examination up to and including the eleventh day of fever. On the twelfth day and each succeeding day none could be found and careful examination of each organ on autopsy has failed to declare them *Theileria parva* were present in considerable number at death—about 60% corpuscles invaded—and for ten days previous.

VI. EXPERIMENTAL TREATMENT.

Despite the absolute failure of any curative or preventive agent in South Africa, it was deemed advisable that certain experiments should be conducted here, for, as will be seen later, there is no doubt but that in some cattle there exists a considerable amount of immunity, and it might be possible in this country to augment this artificially.

Trypanblau.—The success of Trypanblau in the treatment of canine piroplasmosis necessitated its trial in this disease, though Nuttall and Hadwen had already indicated that the action of the drug on *Theileria* was not the same as on *Piroplasma.*

1. Experimental animal No. 51 received intravenously 150.0 c. c. of a 1% solution of Trypanblau=1.5 grms., on the 9th day after exposure, *i. e.* about three days before the temperature might be expected to rise. The official rise occurred on the 15th day, and the temperature remained elevated to death on the 23rd day. Koch's bodies appeared on the fourth day of disease, and remained present, though in comparatively small numbers, to death. On the third day of fever a further dose of 2.0 grms., in 1% solution, was administered intravenously. This did not appear to have any influence on the disease.

One point worthy of note in this animal is that *Theileria* were very scanty throughout. They were first observed on the seventh day of fever, and on the day of death, the 9th day, only about 5% of the corpuscles were invaded.

2. Animal No. 58 had received three injections of serum from a recovered animal (see later). The official rise occurred on the 14th day after exposure, and Koch's bodies were present the same day. On the following day, *i. e.* the second of disease, 1.5 grm. of Trypanblau (in 1% solution) was injected intravenously. This produced but slight and transitory modification in the temperature curve, and on the seventh day of disease a further dose of 2.0 grms. in 1% solution was given intravenously. With this dose there was a sudden fall in the temperature, which remained only slightly above normal for the next seven days. The general state of

the animal, however, rather declined, and Koch's bodies and *Theileria* were very numerous, the latter increasing in number daily. It was necessary to destroy this ox for other experiments on the 14th day of disease, but from its appearance I should suggest that death would have resulted within a few days.

3. Animal No. 55. The object of this experiment was to note the effect of Trypanblau administered on the day of the official rise.

The morning temperature of this ox, which had never exceeded 101.6°F. (38.6°C.), rose on the 15th day to 103.2° (39.6°C.) and 160 c. c. of a 1% solution= 1.6 grms. of trypanblau was injected intravenously. When this amount had been reached the animal ceased breathing, and artificial respiration for several minutes was necessary to restore him to consciousness. That evening the temperature registered 106.4° (41.3°C.), and two evenings later it reached 105.8° (41°C.). Since the day of injection the morning temperature has remained normal, and the animal is alive and perfectly normal in appearance at the time of writing.

Koch's bodies have never been seen, but small piroplasma, probably *P. mutans*, were demonstrated (about 1 to 5 fields stained film) on the seventh day after injection.

Trypanrot.—An aniline dye allied to Trypanblau, introduced by Ehrlich in 1904 for the treatment of trypanosomiasis. It has been used by Nuttall and Hadwen with success in the treatment of canine piroplasmosis, but did not appear so efficacious as the blue dye, and was more irritating.

Animal No. 56. The temperature rose on the 14th day after exposure, and Koch's bodies were demonstrated three days later. On the 8th day of disease 100 c. c. of a 1% solution of trypanrot was injected intravenously. The temperature suddenly fell to normal the following morning, but again rose, and on the tenth day of disease 2.0 grms. of trypanrot, in 1% solution, was given intravenously. The second dose had no beneficial influence on the temperature which continued much elevated to death on the 26th day from exposure, the 13th day of disease. Koch's bodies were very numerous from the day of detection up to death, and *Theileria* increased in numbers progressively, though they appeared much more difficult to stain than is normal.

Experiments with Serum of a recovered Ox, "J".—The animal bled for this purpose was one which I was informed had suffered from Coast Fever at the end of July 1909—positive diagnosis by Koch's bodies. After recovery he was purchased, and early in February, 1910 he was re-exposed to infection on the experimental area. There has been no disturbance in his temperature and he remains in perfect health.

1. To note the effect of Serum "J" injected during the course of the disease.

Case No. 50. The official rise occurred on the 15th day from exposure. On the 17th day 100.0 c. c. Serum "J" was injected intravenously and the temperature remaining elevated a further 100.0 c. c. was injected on the 7th day of disease, and again on the 16th day from the rise in temperature. Koch's bodies appeared on the day after the first injection, and remained present for eight days, after which none were demonstrated.

Theileria were present, about 60%, at death on the 34th day.

This case has been referred to previously (p. 18).

2. To note the effect of Serum "J" injected prior to exposure.

(*a*) Animal No. 57. Received 100.0 c.c. Serum "J" subcutaneously on March 27th, and was exposed to infection 24 hours later. On the 14th day from exposure the morning temperature reached 103°, and on the three following days was 103.5°, 104.6° and 104.8°. On the 18th day it was again 103°, on the 19th and successive days it registered 102° or below, and has remained normal since. Koch's bodies were present on the 17th, 18th and 19th days, since when they have not been seen.

(*b*) Animal No. 58. Received 50.0 c.c. Serum "J" subcutaneously on March 27th and was exposed 24 hours later. On the 7th and 14th days a further dose of 50 c.c. was given subcutaneously.

The temperature became irregular from the 10th day after exposure, the "official" rise occurring on the 14th day, and Koch's bodies were found present.

As previously recorded (p. 18) this animal was subjected to treatment with trypanblau. Koch's bodies continued to be present in the glands and *Theileria* increased in number in the blood. He was destroyed on the 14th day of disease, the 27th after exposure. Death would probably have resulted within a few days.

SYNOPSIS.

Of the ten half-bred animals exposed for these treatment experiments, three are alive two hundred days later. One (No. 52) never showed any symptoms of the disease; one received trypanblau on the day of the "official" rise and recovered. In this case it is not known with certainty that the temperature rise was due to Coast Fever, though its occurrence on the 15th day is strongly suspicious. The third had received serum of a recovered animal a short time prior to exposure; it acquired the disease, as shown by the finding of Koch's bodies, and recovered.

Other experiments have been instituted to control these reactions, but it is most probable that they are due rather to a natural and inherited resistance, such as is observed in some native stock, than to any special influence on the part of the serum or trypanblau, both of which failed completely in other cases when administered under closely similar circumstances.

VII. IMMUNITY IN NATIVE CATTLE.

In the last two Annual Reports of the Chief Veterinary Officer attention is drawn to several instances where, in an outbreak of East Coast Fever, native bred cattle have apparently shown a marked insusceptibility towards the disease; on the other hand we have an instance such as that which occurred on Mr. Russell's farm already quoted; here 121 head died out of a herd of 180, and it is extremely probable that the remaining 59 were only saved by being passed through a temperature camp. During my stay at Kamiti an outbreak of disease amongst calves occurred on the adjoining land of Mr. H. Heatley. This was shown to be East Coast Fever and was suppressed by passing all the animals—roughly 150 cows and 150 calves—through a temperature camp, with a loss of 15 calves only. This is the third outbreak that has occurred on Mr. Heatley's farm, and enquiry elicited the information that in each the disease originated in calves, only in the first did any older animals become infected, and these were all half or quarter-bred stock. The original herd of native cows has never suffered.

Two native cows, the one from Kisii, the other from Nandi country, had already been exposed together with their half-bred calves, on Mr. Russell's infected area. The calves (see p. 10) contracted the disease and died on the 28th and 29th days; the adults have shown no reaction whatsoever, and are alive five months after exposure.

Thirty head of native Kikuyu oxen, the history of which is rather obscure, saving that they had at some time been exposed to infection and had been passed through temperature camps, were exposed to the infected land early in February. The infectivity has been proved by the exposure of four separate lots of susceptible stock. These thirty head have manifested no reaction up to five months later. It appeared desirable to ascertain whether the immunity in these animals could be broken down. Unfortunately facilities were not available for them to be heavily worked in order to reduce their condition, but three were selected and on each of four successive weeks between 3,000 to 5,000 c.c. of blood was removed. That this method had any great effect in lowering the animal's vitality, from a disease resisting point of view, may be questioned; it was not sufficient to break down the resistance to Coast Fever. A similar experiment combining severe exercise and exposure to adverse climatic conditions would be of value.

Six head of cattle, collected in the Kisii country, and which had for the past year worked at Njoro, an area which is regarded as perfectly, clean of Coast Fever, were exposed in April. Up to June 30th none of these animals had shown any reaction. An additional ten animals from Kisii are now being exposed.

Our experiments in this direction are too limited for any sound conclusions to be drawn, but it would certainly appear possible that animals taken from some parts of the Kisii country have a marked resistance, if not an immunity towards Coast Fever. Circumstantial evidence lends colour to the view that Nandi cattle may also possess a resistance. To what cause this insusceptibility is due, still remains obscure. We must, however bear in mind that Bruce, Hammerton, Bateman and Mackie have described the occurrence of Koch's bodies in Kampala, Uganda, where they are associated with a disease, Amakebe, which is held to kill off about 50% of the affected calves. If this disease be Coast Fever, the survivors would possess an immunity.

This question is of considerable importance to the stock owners of the Protectorate, and especially to those in the Nairobi-Kyambu area; it is hoped that the distribution of the immune stock and some of the causes of the immunity may be ascertained within the ensuing year.

APPENDIX III.

Coccidiosis.

In the last Annual Report, Stordy describes under the name Gastro-enteritis a disease which in some cases closely resembles Rinderpest. His endeavours to reproduce it by inoculation and by contact experiments at Nakuru in January 1909 were without success. In August of 1909 Theiler, when on a visit to this Protectorate, was struck by the clinical and pathological resemblance between this new disease and Rinderpest and repeated Stordy's experiments. In a paper which appeared in the *Transvaal Agricultural Journal* (Vol. viii, 1910, p. 195) he confirms the non-identity of the two.

Shortly after my arrival I proceeded to Meru, to the north of Mount Kenya, and found there a disease, which, on clinical and pathological grounds, was similar to Rinderpest. It appeared to have travelled from the West and at the time of my visit it was localised to the country north of Meru itself. Having regard to the information that "Gastro-enteritis", compared with Rinderpest, was very slow spreading, suspicions were aroused and it was eventually shown that in all the very severe cases, and most of the sub-acute, coccidia were present on examination during life. Most of the severe cases were slaughtered and an autopsy obtained. The lesions present were stomatitis (in some cases), some congestion of the abomasum, and even gastritis and acute hyperæmia or inflammation of the intestine from duodenum to rectum; the intensity of the lesions in the several parts varying in the different animals.

Coccidia belong to the highly important group of disease-producing protozoa, their effect in some animals is well known, and they are also responsible for a well recognised disease of cattle in parts of Switzerland where it is responsible for pathological changes similar to those observed at Meru.

It being established that a disease-producing protozoa was associated with the Meru epidemic, it remained to ascertain whether in the widely distributed Gastro-enteritis the same parasite was present, (such an organism being quite in accord with the symptoms as detailed, and the non-inoculability of the disease by means of blood.)

Through the courtesy of the Provincial Commissioner, Kisumu, an area which might be supposed to be heavily infected was obtained there; six young bullocks (Nos. 1 to 6) were purchased from a farm which had not suffered from this disease, and these were exposed on the area on October 16th, 1909.

These experiments had been designed prior to the discovery of Coccidia, and their object was to prove whether the disease could be induced in clean animals, and if so, whether it was by contact, by means of fodder and water, or through the agency of air.

Experiment A.—To ascertain whether animals from a clean farm (supposedly susceptible) could acquire the disease when in contact with the sick, and when grazing off land thoroughly contaminated by sick and dead stock, Two yearling oxen—Nos. 1 and 2—from Elmenteita were exposed to the area on October 16th, 1909. This area was believed to be contaminated, but in addition all the sick animals it was possible to obtain in the locality —about 20—were herded on the same land, slept in the same yard and, on death, the post-mortem was performed in a manner to disseminate infection as much as possible.

No. 1, showed a rise in temperature on the 7th and 10th day after exposure, and on the 8th day was disinclined to feed. No other symptoms were noticed. The temperature returned to normal, but again commenced to rise on the nineteenth day. This reaction lasted for fifteen days, the thermometer registering 103° to 105° as a morning temperature. On the twenty sixth day the animal showed all the symptoms of the disease, and he died on the thirty fourth. Coccidia were present in the fæces from the twenty fifth day up to death.

No. 2, showed no indication of sickness until the twenty fourth day after exposure, when the temperature became slightly raised (about 102° in the morning). On the twenty ninth it was markedly elevated (105° at 7 a.m.) and it remained as a pronounced reaction until the thirty seventh day, when it returned to about normal. From the thirty fourth to the thirty seventh day the animal manifested symptoms of Gastro-enteritis, viz: discharge from eyes and nose, diarrhœa and some hyperæmia of the oral mucous membrane. Coccidia were first seen on the thirty fifth day and were found present up to death on the forty second day. In this case it was hoped that recovery was effected; from the fall in temperature the animal commenced to feed again and acquired a bright outlook. He was found dead in the yard in the morning, and the post-mortem shewed that he had been killed—probably by a herd boy who had hopes of the meat.

Experiment B.—To ascertain whether clean animals which were muzzled and fed off imported fodder and boiled water, could acquire the disease from cohabitation with the sick, and by walking over the contaminated area. Two yearling animals (Nos. 3 and 4) were exposed on October 15th, 1909. They were fitted with leather muzzles six inches deep, having holes an inch in diameter four inches from the bottom to allow ingress of air. These muzzles were worn by day and by night, and were only removed when the animals were brought into a clean yard to be fed and watered at 8 a.m. and 5 p.m. On no occasion are they known to have been removed by intention or accident except at these hours. The fodder consisted of mealie stalks imported from the Government Farm at Kabete (Nairobi) and all water supplied was boiled.

No. 3.—Up to the evening of the nineteenth day the temperature remained practically normal. From then until the twenty eighth day after exposure it was markedly elevated, and from the twenty eighth to the thirty seventh the daily variation was greater than normal, though not markedly elevated. Shortly after the initial rise in temperature, the animal showed all signs of fever, dull roughened coat with dry muzzle and slight constipation. On the evening of the fifth day of disease, during which period only these febrile signs had been present, the faeces were much softer and the following morning they were quite diarrhoeic. At this time there was more dulness and disinclination to feed, excessive discharge from the eyes and an increased amount of mucous in and around the nostrils. On the evening of November 10th, the twenty sixth day after exposure, and the seventh from the initial rise in temperature, the condition of this animal was typical of the disease. Great weakness, hardly able to rise, complete loss of appetite, the eyelids puffy and surrounded by dried scales of discharge which had also scalded the cheek, the hair rough and the skin rather hidebound, the abdomen was tucked up and painful on manipulation : a considerable loss in condition had taken place : the dung was mainly mucus and blood, while the tail and thighs were scalded by the excreta : there was some saliva hanging from the mouth and two small ulcers were present on the gums of the upper jaw. For the next two days the same extreme condition was maintained, but from the third after the fall in temperature a slight improvement became visible. There was increased appetite and vigour, and an attempt to chew the cud : the fæces became more set, though blood and mucous were present for another five days, after which the animal appeared to have completely recovered.

Towards the crisis a dry scaly eruption of the skin appeared on the neck and shoulders.

No.4—A slight rise in temperature—103°—lasting for one day, occurred on the sixth day after exposure; with this exception it remained normal until the twentieth day when a pronounced reaction—104° to 106°), lasting for three days was shown. It fell to normal for two days, when it again rose for a reaction lasting thirteen days. Typical symptoms of the disease were present until the thirtieth day from exposure, but for some days previous there had been excessive lachrymation and slight rhinitis. In this animal Coccidia were only seen on one examination, on the twenty fifth day: merozoites only were present. This animal is still alive and was subsequently inoculated with Rinderpest: like No. 3 a well marked reaction followed the injection of virulent blood.

Experiment C.—To acertain whether clean animals living in a clean yard and which were isolated from actual contact with the sick by means of a fence, but which were fed with grass cut on the contaminated area and received unboiled water from the same source as the others (Lake Victoria), could contract the disease by these means, or by the indirect contagion through air, herdsmen, etc.

Two yearling bullocks (Nos. 5 and 6) from the same farm as 1 to 4 were placed in the clean yard on October 15th 1909. The grass was cut off the pasture used by the rest of the animals—naturally sick and experimentals—and they were given water drawn from the usual cattle watering place on Lake Victoria.

No. 5 died on the twelfth day of the experiment, without showing any sign of this disease, or any disturbance in temperature.

No. 6 was under observation at Kisumu for over forty days, and since then at Nairobi and has not shown any rise in temperature nor other signs of sickness.

If these experiments be accepted, it is evident that healthy animals may show signs of infection after contact or exposure on the land in about three weeks. Every affected animal showed Coccidia on examination during life, and these Coccidia only appeared in the fæces a few days after the initial sign of sickness.

In animals Nos. 1 and 2 which died, merozoites were present in great numbers—npwards of one hundred being often seen in a single microscopic field. (Zeiss DD, occ. IV). In No. 4, which recovered rapidly, they were only seen at one examination; it would appeared that the condition of the animal's body was antagonistic to the multiplication of merozoites, as a result of which, invasion of new cells was checked and recovery effected. In No. 3, merozoite infection lasted for several days and was followed by a period of oocyst formation.

I had an opportunity of examining at Kisumu about twenty cases which a few months before had shown acute signs of "Gastro-enteritis". The animals were all old transport oxen, and the symptoms shown were those of trypanosomiasis. Emaciation and anæmia were the outstanding symptoms, together with a chronic diarrhœa. In nearly all these cases large numbers of oocysts were found in the fæces. On autopsy no definite lesions were discernible save a general anæmic condition and some petechial points especially in the rectum and colon. In scrapings of these two organs oocysts and earlier forms of the sporogony cycle have been seen. The observations possible up to this date indicate that if an animal acquire coccidial infection it may recur without manifesting any definite symptoms; if the body conditions are suitable a very active multiplication of the asexual merozoites may occur. In this case dysenteric symptoms are shown and variable numbers of merozoites are passed in the fæces. Death may take place at this period possibly as a result of debility and fever, possibly due to a toxin elaborated by the coccidia themselves (of this we have yet no direct evidence) or equally possible, as a result of a secondary infection through the wounds already produced in the intestinal canal by the coccidia. Should the animal recover, the merozoite formation and all other developement may cease spontaneously, or, and this appears the more frequent, schizogony gives place to sporogony and the formation of oocysts to enable the species to continue. The duration of oocysts formation may be short in the case of a rapid recovery, in the other cases, which amount almost to the chronic form, there is just that amount of development possible to allow of a continuous formation of oocysts, and their expulsion with the fæces.

I am quite unable to offer any explanation why the animals of Experiment B, developed an infection. The only known method is through the mouth and this should not have occurred in these animals. Again, if the grass and water are the sole means of propagation, Experiment C should also have been successful. It was necessary to repeat these experiments under similar but more rigorous conditions.

The same area was employed, but no sick animals were placed on as contacts. The experimental cattle were all yearling bullocks coming from another clean farm also at Elmenteita and they arrived at Kisumu on December 19th, 1909 and were placed on the area at once.

Experiment A.—Nos. 7 and 8 acted as controls, as also did Nos. 15, 16, 17, 18, 19, 20, 21 and 22, though these last mentioned were submitted to preventative medicinal treatment.

With the exception of No. 15 all these animals showed a marked temperature reaction (106° to 107°) lasting for four to six days, commencing between the 15th and 20th day after exposure. Beyond the temperature no symptoms of sickness were manifested, and examinations of the blood and gland juice were wholly negative.

Experiment B.—Nos. 12, 13 and 14, as in the previous experiment, were muzzled: the muzzles were, however, deepened, and greater care was taken to see that the imported food (in this case hay cut on the farm from which these animals came) was kept from contact with the soiled ground. The water, too was kept at boiling point for an hour.

No. 12 developed a reaction (107.6°) commencing on the 19th day, No. 13 on the 17th; No. 14 did not show such a definite reaction, but its temperature was very irregular for some days commencing on the 22nd day after exposure. In Nos. 12 and 13 the reaction lasted four and five days respectively. No other symptoms were manifested, and examination of blood and gland for other parasites was negative.

Experiment C.—Nos. 9, 10 and 11 were kept in the clean yard and fed with grass cut off the area.

Up to February 10th (52nd day from exposure) these animals showed no temperature reaction, nor any other symptoms of disease. On this date they were liberated on to the area with the other cattle, the reaction in which had been finished for a month. On the 16th and 17th day after liberation the temperature in each case rose, and a reaction similar to that seen in the other animals occurred, lasting for six to eight days. Beyond this temperature reaction no other symptoms were manifested.

As a result of these experiments it is obvious that the area employed carried some agent capable of giving rise to a distinct temperature reaction some sixteen days after exposure, and that this agent was not readily carried in cut grass, by herdsmen or in the air. On the other hand it was in a form which attacked muzzled animals. The reaction itself is, I believe, in accord with what takes place in a coccidial infection and the lack of other symptoms may be ascribed to a resistance of the animals. It is, however, impossible at the present time to explain why the muzzled animals should acquire the disease. The only known way of infection is through the alimentary canal, and this mode should have been eliminated. These experiments cannot be accepted as throwing light upon the manner in which the disease is spread; they, however, serve as evidence regarding the pathogenicity of the Coccidia. An alternate view might be held, viz:—that these animals harbour coccidia without it producing any outward effects; and that exposure to some unknown agent at Kisumu merely stimulated it to increased activity. This is possible, but I would wish to emphasise that this hypothetical stimulating agent cannot have been Rinderpest, to which the symptoms manifested show such similarity.

In the first place the incubative period in every instance was at least sixteen days, whether the animals were in actual contact with the sick or whether they were merely exposed to an area which a month before had carried the sick. In rinderpest the incubative period is usually only five or six days, and rarely as much as ten, Secondly, animals which have suffered from Coccidiosis, and from the temperature reaction, are still susceptible to rinderpest.

Animals Nos. 7, 8, 9, 10, 11, 12, 13, 14, 17, 18, 19 and 20 were removed from Kisumu in March and placed on an area believed to be contaminated with coccidia at Naivasha. From the time of their arrival up to May 10th, 1910, they remained normal. On May 10th, 11th and 12th the temperature of every animal rose suddenly and a pronounced reaction commenced. Symptoms of rinderpest appeared a few days later—extensive stomatitis and ulceration of the lips and gums, diarrhœa, etc., and Nos. 8, 11, 13, 17, 18 and 19 died. Repeated examinations were made during life, and on post-mortem, without demonstrating any coccidia.

On April 14th four new animals Nos. 59, 60, 61 and 62 had been added as controls to prove the coccidial infectivity of the land, and on April 5th an Eland, for which we are indebted to Lord Delamere, had also been exposed. All these animals became attacked simultaneously with the others, viz. May 10th to 12th, and 59 and 61 succumbed.

The disease was introduced among these animals by a mob of trading stock which was found affected between Nakuru and Naivasha, and which was put on to the area which was being utilised for experiments on May 3rd, seven days before the first symptoms were shown by the Kisumu animals.

That this newly introduced disease was rinderpest, is further shown by the subsequent inoculation of Nos. 7, 9 and 12 with 200·0 c. c. of virulent blood from an undoubted case. The reaction which followed was of the mild type, such as results in an already immunised animal, and was unaccompanied by any symptoms.

Of the animals in which coccidia have been positively diagnosed, only Nos. 3 and 4 are at present available.

On July 7th No. 4 was inoculated with 2 c. c. of virulent blood from an experimental case. The temperature reaction commenced on the 9th day, and the animal underwent a typical attack of rinderpest with the symptoms of stomatitis, ulceration of the gums and marked diarrhœa.

No. 3 was inoculated on June 6th with 5 c. c. virulent blood of No. 87. The reaction commenced on the fourth day and lasted five days, during which time the symptoms were typical of the disease.

I have not yet had an opportunity of conducting any inoculations in this territory with the blood of animals suffering from Coccidiosis; it is however, highly probable that this was the disease from which the negative inoculations made by Stordy and Theiler were performed.

In German East Africa a condition is recognised under the name of "malignant catarrhal fever," the symptoms of which are stated to be identical. Lichtenheld, the Chief Veterinary Officer, informed Stordy that from such cases he had made over 50 inoculations, all of which had been negative.

I visited the Island of Ukerewe in February to study a prevailing disease which simulated "Gastro-enteritis". I was able to demonstrate Coccidia in all post-mortem examinations, and in many cases during life. Through the kindness of Dr. Sommerfeldt, the Acting Chief Veterinary Officer, and Mr. Probst, the Veterinary Officer in charge, I was able to carry out some inoculations, from typical cases, into cattle which they considered should be susceptible. The inoculations with blood and spleen were negative to the 14th day, when the animals were destroyed. On post-mortem no lesions recognisable as due to the inoculation were discovered. One animal, whose eyes, nose and mouth had been vigorously rubbed with the discharge from these passages in a sick calf, developed a temperature reaction on the seventh day. He was destroyed and on autopsy showed a marked pharyngitis.

The failure of the blood inoculations, however, inclines me to the belief that the disease I then saw was not Rinderpest. The results of Mr. Probst's previous inoculations, though, lend colour to the view that possibly both conditions—Coccidiosis and Rinderpest—are present in different areas much as we now find is the case here. Further points with regard to the distribution of these two diseases are discussed under the head of Rinderpest (Appendix IV).

THE PARASITE OF THIS DISEASE.

Owing to continued absence on tour, and the lack of laboratory facilities, no systematic work on the structure or development of the Coccidium has been practicable. Such forms as have been observed are in accord with *Eimeria stiedæ* Lind., and with this species I associate the East African form, pending further investigations.

In by far the majority of my examinations only merozoites have been encountered in the fæces and on autopsy. Oocysts have been seen in chronically affected animals and in some of those cases which had recently recovered.

In the dysenteric excreta of an acutely sick animal, and in the scrapings of the inflamed rectum on autopsy, merozoites, either free or arranged like the quarters of an orange within the epithelial cells, are seen in variable numbers. Usually they are very numerous, upwards of a hundred to a field being often present in a fresh coverglass preparation mounted in saline solution. When examined in this manner the merozoites appear as comma-shaped bodies of a pale straw colour, capable of flexion and extension, but of very limited progression. In the fresh state they measure about 11 to 14 microns in length (some, however, appear as short as 6 or 8 microns), and from 1.5 to 2.5 microns at the widest part, which lies at about 33% of the length, the upper third is rounded at the extremity and the lower two thirds taper to a blunt point. There appear two types of merozoites, the one longer and finer than the other; the significance of this has not yet been ascertained here, but they may represent early sexual dimorphism.

The oocysts range from 14 to 20 microns in length, by 12 to 16 microns in breadth (average 17 by 15 microns); they are clear and transparent with a definite contour and finely granular protoplasmic contents frequently aggregated towards the pole, opposite to the micropyle.

Observations on the development of sporoblasts within the expelled oocysts are now being made, and it is hoped some results may be obtained in artificial infection during the year.

APPENDIX IV.

Rinderpest.

In the absence of a better term, the word "Rinderpest" is employed to signify the inoculable disease resembling Coccidiosis which has already been mentioned. There is a great probability that the word is used correctly, though final proofs are still wanting.

Reports were received in May that a severe outbreak of gastro-enteritis had occurred at Njoro, and along the Caravan Road through Nakuru to the Quarantine Station at Naivasha. Inability to detect coccidia in the material submitted, and the unusual severity of the outbreak, necessitated further enquiries, and it was proved that a small quantity of blood taken from a sick animal would reproduce the disease in a susceptible bovine.

TO DEMONSTRATE THE INFECTIVITY OF BLOOD.

Three half-bred bulls bred and reared on the Government Farm Naivasha arrived at Njoro on the 18th May. To avoid any chance of infection by coccidia en route, they were taken to the train on a cart, and during the experiment were kept in the railway cattle truck and fed upon imported fodder.

No. 84—was inoculated on May 18th with 100 c.c. blood stained serum from a case on Mr. S.'s farm. The reaction commenced on the sixth day and lasted until the thirteenth. During this period the temperature was only twice below 105° F. Marked discharge from the eyes and nose was present on third day of fever, and inflammation of the incisor gums two days later. On the 14th day the temperature fell, and the animal recovered.

No. 85—was inoculated on May 20th with 250 c. c. citrated blood drawn during life from a case "A," the property of Mr. C. The reaction commenced on the fourth day (temperature 104.3° at 8 a. m.) and lasted for seven days. On the 11th day from inoculation the animal was moribund and was destroyed.

The symptoms manifested by this animal were acute conjunctivitis, rhinitis and stomatitis with ulceration on the lips, and marked diarrhœa containing blood. On post-mortem two circumscribed ulcers were seen on the dorsum of the tongue and a diphtheritic catarrh on the hard-palate and in the pharynx. The pyloric extremity of the abomasum was markedly inflamed but there was no ulceration. The small intestine was hyperæmic throughout; Peyer's patches were raised and coated with an adherent diphtheritic mass. The large intestine was hyperæmic, with hæmorrhages in the colon: the rectal lesions were less marked: the intestinal contents were very fœtid and blood stained. The liver was enlarged and the gall-bladder contained 150.0 c. c. green coloured bile.

No. 86—was inoculated on the same day and in like manner to No. 85. On the morning of the fourth day the temperature was 105° F and the reaction continued for six days: to the day of death, 23rd after inoculation, the temperature remained irregular. Clinical symptoms—nasal and ocular discharge—were noticeable on the 8th day after inoculation. Towards the end of the febrile reaction they were marked. A pronounced rhinitis and severe ulceration inside the lips and on the gums. Diarrhœa was constant from the sixth day up to death; blood and mucus were also present in the excreta.

On post-mortem, the ulceration in the mouth had healed. There were two resolving ulcers on the pyloric ring. In the small intestine there was generalised, but not pronounced, hyperæmia: Peyers patches showed small suppurating points. The large intestine, and especially the rectum was markedly inflamed. The liver was friable and the gall-bladder contained about 250 c.c. rather viscid green bile.

These experiments demonstrated the inoculability of this disease with large quantities of blood. It remained to prove that a small amount would equally convey the infection and that the disease could be transmitted indirectly by fomites.

No. 87. Half-bred bull from the Government Farm. Inoculated on May 30th with 2.0 c.c. blood direct from No. 85 just destroyed. The reaction commenced on the 5th day and lasted until the 13th. From this until the 16th the temperature shows great diurnal variation — from 101° or 102° in the morning to 107° to 107.8° at night. The animal died on the night of the 16th day from inoculation. The symptoms and post-mortem appearance were similar to those noted in cases Nos. 85 and 86— viz., discharge from eyes and nose, ulceration of the gums and profuse diarrhœa containing blood and mucus. Acute gastritis with ulceration of the pyloric extremity of the abomasum: hyperæmia and inflammation of the intestine.

No. 88. Another animal from Government Farm to demonstrate contagion. On May 30th cases 84 and 86—twelfth and tenth days from inoculation—were removed from the cattle truck in which they had lived since leaving Naivasha, and the body of No. 85 was also taken away. The soiled litter was allowed to remain and No. 88 was placed in the truck and fed upon imported fodder. The temperature reaction commenced on the fifth to sixth days after exposure within the truck, and the animal died on the 12th day. The symptoms and post-mortem lesions were typical of the disease.

Two other experiments support the view of contagion.

No. 94. A half-bred animal from Government Farm—was placed in contact with the sick and inoculated animals on June 5th. On the 7th to 8th days after exposure the temperature rose and a marked reaction, accompanied by pronounced clinical symptoms, followed. There is every probability that this animal would have succumbed. On the 15th day, when the case was, indeed, severely affected, a dose of Rinderpest serum was given sub-cutaneously and the animal recovered.

No. 100. A fifteen months old native bullock—bred on a farm at Elmenteita, was exposed in like manner to No. 94 on June 5th. The temperature reaction commenced on the 14th day (June 18th and 19th) and the animal suffered from a typical attack of the disease from which it recovered.

TO NOTE THE EFFECT OF ANTI-RINDERPEST SERUM.

This serum was obtained from the Transvaal Government some years ago when there was fear of Rinderpest entering from the North.

HALF-BRED ANIMALS.

Six two year old heifers—Nos. 89 to 94—were obtained from the Naivasha, Government Farm, and were tested on June 5th 1910.

No. 89—received 100 c.c. Pretoria Serum No. 60, plus 2.0 c.c. virulent blood from No. 87. From the 4th to the 7th day after inoculation the evening temperature was elevated—105° to 106°F.—the morning remained practically normal, and no symptoms were shown.

No. 90—received 50.0 c.c. Pretoria Serum No. 60, plus 2.0 c.c. virulent blood of No. 87. The temperature rose on the fourth day, and a marked reaction lasting for six days followed. All symptoms of the disease were shown by this animal. From the 12th day after injection the temperature remained normal and recovery was rapid.

No. 91—received 20.0 c.c. Pretoria Serum No. 60, plus 2.0 c.c. virulent blood of No. 87. The reaction commenced on the 4th day and lasted 10 days. The animal showed all symptoms of the disease and appeared on the verge of death about the 8th day of reaction, but has made a slow recovery.

Control Animal No. 93—received 2.0 c.c. virulent blood of No. 87 and no serum. The reaction commenced on the 4th day. On the 8th all symptoms of Rinderpest were present, and death took place on the night of the 11th.

NATIVE BRED ANIMALS.

Six yearling to fifteen months bullocks—Nos. 95 to 100—were obtained from a clean farm at Elmenteita.

Control Animals Nos. 95, 96 and 97.—These animals received varying amounts of virulent blood of No. 87 to test the susceptibility of the native stock towards the virus. The reaction commenced in each case on the third and fourth day, and lasted six and nine days in the animals which recovered and nine days in the one which died. All symptoms of Rinderpest were shown, though in the one (No. 97, reaction six days), which only received 2.0 c.c. virulent blood, the characteristic lesions were limited to a marked inflammation of the alveoli, and did not proceed to ulceration as in the other two cases.

Serum Testing—*No. 98* received 50.0 c.c. Pretoria Serum No. 60 and 2.0 c.c. virulent blood of 87. There was a slight reaction lasting six days commencing from the 5th after inoculation. This reaction was practically confined to the evening temperature, the morning being only slightly elevated on two occasions. The only symptoms shown were slight discharge from the eyes and nose.

No. 99—received 20.0 c.c. Pretoria Serum No. 60, plus 2.0 c.c. virulent blood of No. 87. A reaction lasting six days commenced on the fourth day. It was more marked than in the case of No. 98 but no more severe symptoms were manifested.

SERUM AS A CURATIVE AGENT.

No. 92—half-bred heifer—was inoculated with 2.0 c.c. virulent blood of No. 87 on June 5th. The temperature became irregular on the 4th and 5th day. On the 5th day 250.0 c c. Pretoria Serum No. 60 was given subcutaneously. The reaction lasted five days but the temperature was irregular throughout. Symptoms of disease appeared on the seventh day and on the eighth there was pronounced ulceration of the gums. The temperature remained irregular for a week after the primary reaction was over, but the animal recovered.

No. 94—half-bred heifer—This animal was placed in contact with the sick on June 5th. The first rise in temperature occurred on the seventh day after exposure and a pronounced reaction had lasted for nine days when 250.0 c.c. Pretoria Serum No. 60 was injected subcutaneously. On the day of serum injection the animal was off feed, very sick and typical of the disease. From its general appearance an unfavourable prognosis would have been justifiable.

With this evidence regarding the value of anti-rinderpest serum before us, I consider it most essential to the interests of the country that its manufacture on a large scale be commenced without delay. The supply of serum which came from the Transvaal has been examined to eliminate the bottles which had gone bad, and the rest has all been mixed and rebottled. This serum, amounting to about 150 litres, is now being tested to ascertain the approximate dose: when tested it will be ready for issue, and in the meantime it is hoped a sufficient supply of local manufacture will be available. There are, roughly, between ten and eleven thousand head of cattle owned by Europeans in this Protectorate. These I consider should be inoculated first, that the results obtained may be an object lesson to the native owners, who might otherwise resent an interference with their stock.

The cost of production and administration for the European owned cattle would not, I think, exceed £1,200, towards which I believe most owers would willingly contribute in the form of a tax on each animal successfully immunised.

As a result of these experiments it is quite obvious that we are dealing with a disease distinct from that studied by Stordy and Theiler, whose inoculating tests were entirely negative. The animals which they employed were drawn from the same herd on the Government Farm at Naivasha. The negative results obtained by them are, therefore, not due to any immunity on the part of the animals. In the 18 inoculations and contact experiments which I have made up to date, not one has failed to give a pronounced reaction, and the animals utilised include cattle of the native breed in addition to the more susceptible half-bred stock.

On page 27 I have cited the case of animals recovered from Coccidiosis acquiring an attack of Rinderpest on inoculation; an attack of the former does not then give an immunity to the latter. This fact is of interest, since the evidence up to the present time is that farms which were affected in 1909 have not suffered again, and that only those which remained healthy last year have been affected. If this view continues to hold good, it would appear very possible that in some areas of the Highlands—Lumbwa to Nakuru—the 1909 disease on many farms was Rinderpest: on others it may have been Coccidiosis, and it is conceivable that such animals were utilised by Stordy and by Theiler.

That the two diseases have been confused is, I think, undeniable: and there is strong evidence that in the Molo district, the disease was not Rinderpest.

Messrs. A's farm is situated fifteen miles from the main road down which the infected trade stock was travelling, and no cattle pass through their farm, which is thus well isolated. On June 30th they took an animal from this farm to Nakuru and brought it back on July 6th. On July 19th this animal was sick with all the symptoms of "Gastro-enteritis." On July 30th the first case occurred in animals which had never left this land and which had had no contact with outside stock, and the disease remained among the herd until the middle of September. The mortality was two cows and seven calves in a total of 80 head attacked.

The owners are unable to ascribe any other reason than the above for the introduction of the disease. The delay of thirteen days between the return of the Nakuru animal and the onset of symptoms is hardly in accord with Rinderpest, or again, is the delay of eleven days from the time when symptoms were first seen in this and in the autochthonons animals. The outbreak is more in accord with Coccidiosis or a disease having a long incubative period.

Whatever degree of immunity may exist in the native bred stock of this country, it would be expected that animals imported from England would have been highly susceptible. On one farm at Molo, this did not appear to be the case towards the prevailing disease.

"Gastro-enteritis" broke out on Mr. C.'s land in March of this year, (1910), and attacked some pure bred imported Devon heifers and calves, which were housed with two Devon imported bulls, an open partition being the only separation. Two of the heifers died, but the bulls never showed any symptom of disease although they were in such close proximity and were attended by the same herdsman.

Similar cases have been recorded by Lord Delamere, by the Hon. R. B. Cole at Njoro, and I believe by other stock owners.

It is however, probable that prior to its detection at Njoro in May, 1910, Rinderpest had existed during the year in East Africa. Sickness appeared at Kericho among the Kisii captured stock about August, 1909. The death rate was not high, but thirty five of these removed to Njoro in September, 1909, have remained unaffected on Mr. S.'s farm where the mortality among the rest was nearly 40%. About the same time as the Kericho outbreak, the disease appeared on the farm of Major C. a few miles away. The death rate there was considerably higher than in any other reported outbreak.

The disease which was investigated at Njoro is held to have reached there from the Rongai River where the Njoro farmers have been in the habit of grazing their cattle. Some of these animals on returning introduced the disease to the rest. The natural incubative period—the time elapsing from the date of the return of animals from the Rongai to that on which the first autochthonous case arose—has been from seven to ten days and the mortality on these farms has been 57% , 24% , 39% , 20% and 48% , considerably higher than in the old "Gastro-enteritis." The results of inoculation with infective blood have already been given.

The present position with regard to our knowledge of these two diseases —Coccidiosis and Rinderpest—is highly unsatisfactory from a stock owner's point of view: it is still more dissatisfying from a scientific standpoint. I have hope that during the ensuing year additional evidence will be collected, and that the new Laboratory accommodation will permit of exact and final experiments being performed to settle some of many points which are at present debated.

I cannot close the reports on this branch of the year's work without giving a word of thanks to Dr. Hannigan, the Stock Inspector in whose charge nearly all the coccidia and rinderpest experimental animals have been during my absence in other parts of the Protectorate. It is a pleasure to record the interest he has taken in the experiments, and the thorough way in which his records have been kept.